Jose R. Romero, Ph.D.

Instructor in Medicine
Harvard Medical School

Associate Physiologist
Division of Endocrinology/Hypertension
Brigham and Women' s Hospital

Research Interests

The main research goal of my laboratory is to define the signal transduction pathways involved with the regulation of cation transport mechanisms across the cell membrane as they affect human cardiovascular disease. The central hypothesis for our research is that cellular cation metabolism plays a major role in the pathophysiology of cardiovascular disease by regulating the production of reactive oxygen species, nitric oxide, and cellular volume. We have used human erythrocytes as an *ex vivo* model of cation transport alterations in the pathophysiology of various cardiovascular diseases. More specifically, our laboratory is interested in the role of sodium and potassium transporters in hypertension and diabetes. In human blood cells (erythrocytes and lymphocytes), we study the renin-angiotensin system and insulin regulation of the sodium, magnesium, calcium, and potassium transport mechanisms. Some of these observations have allowed us to pursue more detailed studies in nucleated cells and to target tissue of these diseases, such as human microvascular endothelial cells and fibroblasts. In these cells, we have been studying the role of cytokines and other vaso-active compounds in cation metabolism to elucidate the signaling pathways that are regulated by these agonists as well as their role in reactive oxygen species, fibrogenesis, and nitric oxide metabolism.

These studies have proven promising and form the basis of our current National Institutes of Health grant and our most recent publication, which looks at the cellular magnesium dysfunction in diabetes and its role in nitric oxide production. Attempts to subdivide the large hypertensive population based on intermediate phenotypes now serve as the basis for genetic studies of pathogenesis, with which we are also currently involved. To this end, and in collaboration with
Dr. Naomi Fisher and Dr. Gordon Williams from our Division, we are currently studying the influence of race on the renin-angiotensin system and on the *in vivo* regulation of K^+ transporters. In addition, the research projects of my laboratory have given various students the opportunity to learn, participate, and expand the scope of their training. Furthermore, because of our expertise in cation metabolism in erythrocyte volume regulation and its role in the pathophysiology of sickle cell disease, we maintain a productive collaboration with Dr. Ronald Nagel from the Montefiore Medical Center, with whom we are studying the *in vivo* role of nitric oxide on the Ca^{2+}-activated K^+ channel and the K^+/Cl^- cotransporter in mice and humans.

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